ORIGINAL ARTICLES

DYSBARIC ILLNESS TREATED AT THE ROYAL ADELAIDE HOSPITAL 1987 A FACTORIAL ANALYSIS

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Introduction

The accepted predispositions to both decompression sickness (DCS) and arterial gas embolism (AGE) in divers are listed in diving medicine texts[1], but supporting data are often anecdotal, and analyses of series of divers with these diseases have caused the significance of some of these predispositions to be questioned[2-4]. The grading of DCS into clinical types I and II does not appear to be predictive of outcome[5,6], perhaps as a result of the remarkable variability seen in the manifestations of this illness[7]. This variability, and the limited data available for the treatment of AGE[8], may explain why the management of these conditions remains controversial. Also, while it is conceded universally that both DCS and AGE can affect the nervous system[3,9-13], agreement on the frequency of neurological involvement[6, 10, 12-15], and on both the frequency and the nature of neurological sequelae[6, 16-21], cannot be reached.

It is clear that the understanding of these illnesses is limited.

During 1987, 64 divers were treated at the Royal Adelaide Hospital Hyperbaric Medicine Unit (RAH HMU) for either DCS or AGE. While this is not a large series, it is nevertheless of interest in that the mean time from the onset of the diver's symptoms and signs to compression in a recompression chamber (RCC) was relatively long, 26.4 hours, and the frequency of incomplete resolution of symptoms and signs at the time of the diver's discharge from the hospital was high, 54.5%. Consequently, a factorial analysis of these diving accidents was performed to determine what factors were associated with a poor outcome. The results are discussed in the context of the controversies listed above.

Clinical Review Period; 1987

Sixty-four divers suffering dysbaric illness, 58 with DCS, and 6 with AGE, were treated by recompression at the RAH HMU. None of these had used gas mixtures other than air. All of the divers were treated in a multiplace RCC, and with one exception they were intravenously rehydrated with crystalloid solutions. Only 2 of the divers with DCS were given intravenous steroids and none intravenous lignocaine. Amongst those with AGE, one diver received both intravenous steroids and lignocaine.

The RAH HMU protocol for treatment of either illness involves initial compression to 2.8 Bar absolute pressure (1 Bar = 1 atmosphere) and administration of 100% oxygen to the casualty. For those being treated within 24 hours of the onset of their disease, if this regimen fails to achieve significant relief, then the diver is changed to either an oxygen-nitrogen or an oxygen-helium mixture (depending on the gas breathed during the dive) and compressed further to either 4 or 6 Bar. Recurrent or persistent symptoms and signs are treated with daily hyperbaric oxygen (HBO) exposures, until either resolution has been achieved or the deficit is considered refractory to treatment.

Of those divers with DCS, 22 presented for treatment within 24 hours. All had their symptoms and signs completely relieved during the initial compression treatment. However, 16 relapsed and required repeat HBO treatments. Repeated treatments were successful in only 6 of these 16 divers, so that 10 left hospital with persistent symptoms and signs.

The remaining 36 divers with DCS (62%) presented for treatment 24 hours or longer after the onset of their illnesses. Despite repeated HBO treatment, 21 of these divers never experienced complete resolution of their symptoms and signs.

Overall only 27 divers with DCS (46.5%) had complete relief during their period of hospitalisation. None of the residual symptoms and signs in the remaining 31 divers with incomplete resolution were present prior to the current episode of DCS. These residual phenomena were predominantly arthralgia (25 divers) and neurological symptoms and/or detectable neurological deficit (8 divers). Two divers had both arthralgia and a neurological deficit.

Only 6 of the 64 divers had AGE. In 2 of these symptoms and signs did not fully resolve.

Data from all divers were recorded on a designated proforma and transferred to a computerised data base (dBase III plus). Data were analysed using Fisher's Exact Probability Test[22].

Results

The data for all divers with DCS are summarised in Table I (p 100) and for those with AGE in Table II (p101).

Those factors that were significantly ($P < 0.05$) associated with incomplete resolution of DCS symptoms and signs, with the calculated level of probability,are listed in Table III. Those factors that were not significantly ($P > 0.05$) associated with incomplete resolution of DCS symptoms and signs, with the calculated level of probability, are listed in Table IV.

TABLE III

FACTORS SIGNIFICANTLY ASSOCIATED WITH INCOMPLETE RESOLUTION OF DCS SYMPTOMS AND SIGNS IN 58 CASES OF DECOMPRESSION SICKNESS

	Factor	Fishers Exact Probability
1.	Previous DCS:	0.012
2.	Compliance with DCIEM tables:	0.050
3.	Abalone divers:	0.003
4.	Number of ascents:	
	1 v more than 1;	0.034
	Less than 3 v 3 or more;	0.045

TABLE IV

FACTORS NOT SIGNIFICANTLY ASSOCIATED WITH INCOMPLETE RESOLUTION OF DCS SYMPTOMS AND SIGNS IN 58 CASES OF DECOMPRESSION SICKNESS

	Factor	Fishers Exact Probability
1.	Age: Less than 40 .v 40 or older;	1.000
2.	Sex:	1.000
3.	Occupation:	
	Manual v sedentary;	0.590
4.	Maximum depth:	
	Less than 10 msw v 10 msw or deeper;	0.116
	Less than 20 msw v 20 msw or deeper;	0.162
	Less than 30 msw v 30 msw or deeper;	0.172
	Less than 40 msw v 40 msw or deeper;	0.694
	Less than 50 msw v 50 msw or deeper;	1.000
5.	Number of divers:	
	1 v more than than 1;	0.782
	Less than 2 v 2 or more;	0.292
	Less than 6 v 6 or more;	0.131
6.	Type of disease	0.773
7.	Time to onset of symptoms:	
	1 hour v more than 1 hour;	0.174
	Less than 6 hours v 6 hours or more;	0.425
8.	Delay prior to compression:	
	Less than 6 hours v 6 hours or more;	0.212
	Less than 12 hours v 12 hours or more;	0.193
	Less than 24 hours v 24 hours or more;	0.420
	Less than 48 hours v 48 hours or more;	0.271
9.	Initial hyperbaric treatment table:	

TABLE I

58 CASES OF DECOMPRESSION SICKNESS

1.	Mean age:		30 years	
2.	Sex:	Males;		46
		Females;		12
3.	Occupation:	Manual;		35
		Sedentary;		23
4.	Activity:	Recreational;		39
		Instructing Recreational		1
		Scientific;		3
		Fishing (eg. abalone);		12
		Commercial;		2
		Dry Chamber dive		1
5.	Previous decompression sickness:		13 (22%)	
6.	Mean experience (one diver had no formal training)		2.17 years	
7.	Mean depth:		25 msw	
8.	Gas mixture:	Air ;		58
		$O2N_2$;		0
		O_2H_e;		0
.	Compliance with DCIEM tables:		18 (31%)	
10.	Decompression tables:	None;		15
		USN;		16
		PADI;		19
		Others (eg. RNPL);		8
		Decompression meters;		0
11.	Repetitive dives:		40 (69%)	
12.	Mean number of dives:			2.9
13.	Mean number of days diving:			2.2
14.	Mean number of ascents:			3.15
15.	Alcohol intake on the same day before the dive:			0
16.	Symptoms precipitated by decompression to altitude:		5 (8.6%)	
17.	Equipment failure:		6 (10.3%)	
18.	Mean time of onset of symptoms and signs:		6.48 hours	
19.	Type of disease:	Type I;		17
		Type II;		41
20.	Diving Emergency Service contact:		41 (70.7%)	
21.	Retrieval to RAH HMU:		43 (74.1%)	
22.	Retrieval by transportable RCC:		30 (51.7%)	
23.	Mean delay from onset of DCS to compression in a RCC		26.4 hours	
24.	Multiple hyperbaric treatments:		44 (75.9%)	
25.	Mean number of hyperbaric treatments:			3.43
26.	Intravenous steroids:		2 (3.4%)	
27.	Intravenous lignocaine:			0
28.	Outcome:	Complete resolution:	27 (46.5%)	
		Incomplete resolution:	31 (54.5%)	

TABLE II

6 CASES OF ARTERIAL GAS EMBOLISM

1.	Mean age:		31.6 years	
2.	Sex:	Males;		
6		Females;		0
3.	Occupation:	Manual;		5
		Sedentary;		1
4.	Activity[1]:	Recreational;		3
		Instructing Recreational;		1
		Scientific;		1
		Fishing (eg. abalone);		0
		Commercial;		1
5.	Previous arterial gas embolism:			0
6.	Mean experience (3 experienced divers and 3 scuba trainees):		3 years	
7.	Mean depth:			25 msw
8.	Gas mixture:	Air;		6
		O_2N_2;		0
		O_2H_e;		0
9.	Compliance with DCIEM tables:		3 (50%)	
10.	Decompression tables:	None;		1
		USN;		2
		PADI;		
		Others (eg. RNPL)		1
		Decompression meters;		0
11.	Repetitive dives:		2 (33.3%)	
12.	Mean number of dives:			2.5
13.	Mean number of days diving:			2.
14.	Mean number of ascents:			1.0
15.	Alcohol intake on the same day before the dive:			0
16.	Symptoms precipitated by decompression to altitude:			0
17.	Equipment failure:		2 (33.3%)	
18.	Mean time of onset of symptoms and signs:		19.8 minutes	
19.	Type of disease:	Neurological;		6
		Cardiological/pulmonary;		3
20.	Diving Emergency Service contact:		3 (50%)	
21.	Retrieval to RAH HMU:		4 (66.7%)	
22.	Retrieval by transportable RCC:		3 (50%)	
23.	Mean delay from onset of AGE to compression in an RCC:		19.98 hours	
24.	Multiple hyperbaric treatments:		6 (100%)	
25.	Mean number of hyperbaric treatments:			2.9
26.	Intravenous steriods:		1 (16.7%)	
27.	Intravenous lignocaine:		1 (16.7%)	
28.	Outcome:	Complete resolution:	4 (66.7%)	
		Incomplete resolution:	2 (33.3%)}	

Discussion

Most reviews of divers with dysbaric illnesses have described early recompression treatment, and have reported high resolution rates[14]. However, the same authors have described persistent arthralgia in some divers despite conventional therapy, and furthermore, the frequency of neurological sequelae varies with the extent of investigations and the intensity of follow-up examinations[6, 16-21]. The natural history of these persistent symptoms and signs is for spontaneous resolution over weeks to months[6,14], and this resolution can occur despite persistent histologically-evident nervous system damage[23]. As such, much of the nerous system recovery may be due to the recruitment of previously uncommitted neurons.

Delay prior to treatment appears to be an important determinant of outcome[3, 9, 24-27]. In a series such as the one reported here, this finding will be obscurred by the earlier onset of fulminant neurological DCS in comparison to that of milder disease[28], and the more likely early presentation for treatment of divers with such severe disease. The important finding in this series is the frequency of incomplete resolution of symptoms and signs. Regardless of the outcome of these residual symptoms and signs, their frequent occurrence enables a factorial analysis to be performed. Such analyses are impossible in those series where the resolution rates are high[14].

Predispositions to DCS

The accepted predispositions to DCS include[1, 29], being female, increasing age, multiple dives, multiple ascents, multiple days diving, diving at altitude, decompression to altitude after diving, ingestion of alcohol, exercise, cold stress, obesity, dehydration, retention of carbon dioxide, physical injury, fatigue and the level of complement protein activity.

The data presented here do not permit detailed discussion of most of these phenomena, because the total diving exposure of the community from which our patients are derived is not known. For example, it is necessary to know the percentage of the total hours of diving attributable to females, before the significance of the 12 female divers (20.7% of the total) with DCS in this series can be determined.

However, some significance can probably be attributed to the following observations: 69% of all of these divers had dive exposures that exceeded the limits of the DCIEM decompression tables[30] These tables were chosen as a reference because they were developed after extensive laboratory and field testing and the associated probabilities of DCS are known[31]. 25.8% of the divers were not using a decompression table of any sort. 69% of the divers developed DCS after a series of repetitive dives. Only 10.3% had their episode of DCS precipitated by equipment failure. The majority of patients (70.7%) had neurological symptoms and signs. And despite 75.9% of these diver patients receiving multiple HBO treatments, 54.5% had incomplete resolution of their symptoms and signs. In addition, 5 divers developed symptoms of DCS only after a decompression to an altitude of more than 300 metres above sea level. In 3 of these episodes, this decompression occurred between 12 and 24 hours after the dive, and in one, more than 24 hours after the dive. DCS occurred in 2 of these 5 divers despite them having done dives that complied with the DCIEM tables[30].

It is reasonable to argue both that the use of any decompression table is probably better than using none at all, and from the data presented here, that recreational divers should use a conservative decompression table such as that produced by DCIEM[30]. The frequency of repetitive diving in this series of DCS is consistent with convention[1], but contrasts with the 1987 experience of the United States of America Divers' Alert Network (DAN), in which 75% of DCS episodes resulted from a single dive[2]. Nevertheless, the observation that repetitive diving is a risk factor for DCS is not surprising, given the demonstrated slow clearance of inert gases from biological systems in comparison to rates of uptake[32], and the observation that should gas phase separation occur during the decompression from a dive, that gas elimination will be even slower on subsequent (repetitive) dives[33].

The data presented here are also in conflict with the 1987 DAN experience in 2 other areas: the absence of alcohol as a risk factor (c.f. DAN; alcohol in 50% of cases of DCS); and the absence of decompression meters as the only controller of decompression (c.f. DAN; 38 cases of DCS in divers using meters in such a fashion)[2]. The former observation is particularly surprising, and the latter may change as the use of decompression meters becomes more widespread in Australia[34].

Although it is claimed by some authors that the majority of divers who develop DCS after a dive where nitrogen is the diluent gas involved will have isolated musculo-skeletal disease[14, 15], 70.7% of divers with DCS in this series had overt neurological symptoms and signs. These data are consistent with those reports that suggest that most DCS incidents will involve the nervous system[10, 12, 13].

Despite multiple hyperbaric treatments, more than half of these divers did not have their symptoms and signs fully resolved before they left hospital. Although this greatly exceeds the anticipated failure-rate for hyperbaric treatment of this disease[14], it is in agreement with the finding that almost half of a series of divers with DCS had abnormal electro-encephalograph (EEG) findings one week after they had completed treatment for DCS[6]. In that series the prevalence of abnormal EEGs fell significantly ($P < 0.0001$) during the subsequent month suggesting that the abnormalities were indeed related to the episode of DCS.

The time-frame of the altitude-precipitated episodes of DCS is a strong argument that divers should not fly within 24 hours of a dive, regardless of the nature of that dive.

Predispositions to AGE

Although not established, it is widely accepted that AGE complicates the decompression of divers because of an increase in airway pressure which can cause gas embolism of the pulmonary veins[9, 35, 36]. Consequently, it is argued that rapid decompression, breath-holding, and pulmonary pathology that can result in air trapping predispose to AGE[1]. However, reviews of patients who have suffered AGE have shown a very low concurrence of overt pulmonary damage (e.g. pneumothorax, mediastinal emphysema, surgical emphysema)[3]; and the pulmonary lesion that typically underlies the evolution of gas emboli in this situation has not been described. In this small series, firm conclusions are not possible, but 2 of these 6 divers performed a free ascent to the surface after equipment failure, and 3 of the 6 were recreational scuba trainees. All divers had neurological manifestations, and 3 had symptoms of chest pain and/or dyspnoea. None of these 3 divers had radiological evidence of pulmonary barotrauma, and their symptoms may have been cardiac in origin. Cardiac symptoms can occur when arterial gas emboli enter the brain stem circulation and so affecting neural control of heart function, by emboli entering the coronary circulation, and the heart chambers, or cardiac function can be indirectly affected by emboli enhancing the release of catecholamines into the systemic circulation[35, 37-40].

The mean time of onset of symptoms was almost 20 minutes after completion of the decompression. This finding conflicts with the convention that the onset of this disease is within 5 minutes of decompression[1], but is in agreement with other reports of delayed presentation[3, 9]. The probability of DCS calculated from their reported dive profiles was less than 1% ($p\ DCS < 0.01$) for each of these divers[31], so an incorrect diagnosis is unlikely. Two of the 6 divers had incomplete resolution of their symptoms and signs despite repeated HBO exposures, but given their delay prior to treatment (mean delay = 19.98 hours) this is not surprising[3, 9, 24, 25, 27]. These small numbers prevent further analysis.

Factors that influence the outcome of patients treated by recompression for DCS

The following were not significantly associated with outcome (complete or incomplete resolution): sex, occupation, increasing age, increasing maximum depth, an increasing number of dives, the type of DCS presentation (I or II)[5], increasing time from decompression to the onset of symptoms, and from the onset of symtoms to compression; the initial hyperbaric treatment used, and the number of hyperbaric treatments. While the insignificance of age is surprising, and it appears from other studies that the more severe the DCS the shorter is the latency before symptoms develop[28]; many of these results can be explained by the probable earlier presentation for treatment of those divers with the more severe forms of DCS, and the more frequent and aggressive treatment of such divers. The similar frequency of incomplete resolution of symptoms and signs for those divers with type I and those with type II DCS can also be explained by this phenomena, but may also be interpreted as supporting the argument that this typing of DCS is not predictive of outcome[6]. Indeed, it is likely that many divers with type I DCS have covert nervous system involvement[6].

In contrast, being a diver whose occupation is collecting abalone, a history of previous DCS, having had a diving exposure that did not comply with the DCIEM decompression tables[30], and an increasing number of ascents (decompressions) were all significantly associated with a poor outcome (incomplete resolution).

The local abalone diving community has been investigated previously[16], and shown to have a high incidence of DCS, and an increased prevalence of hearing loss and dysbaric ostenonecrosis. Twelve abalone divers were treated for DCS in this series, and 11 had incomplete relief. Nine of the 12 abalone divers had a past history of at least one episode of DCS.

The association between a previous history of DCS and poor outcome is highly suggestive that much of the recovery from DCS may be due to phenomena such as neuron recruitment, and requires that a return to diving after an episode of DCS should involve very conservative decompression practises to minimise the risk of further DCS.

The significance of both complying with conservative decompression tables such as those issued by DCIEM[30], and of minimising ascents so as to improve outcome is obvious from these data. Multiple ascents are already known as a risk-factor for DCS[1], but this study shows that not only may multiple ascents increase the probability of a diver developing DCS, but also that dives involving multiple ascents reduce the chances of a good outcome after treatment of any ensuing DCS.

Summary

The effects of treatment in a series of 58 divers with DCS and 6 with AGE, that occurred after dives involving compressed air, are presented. The findings in this series include a predominance of neurological DCS, long delays prior to treatment by recompression, and a poor overall resolution rate. Factorial analysis shows that the chances of a poor response to treatment increase if the diver has a previous history of DCS, if the diver is are an abalone diver; and if the dive profile did not comply with conservative decompression tables such as those of DCIEM[30], or if it involved multiple ascents.

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DIVING ACCIDENTS WHAT IS THE MAGNITUDE OF THE PROBLEM IN NEW ZEALAND ?

Allan F.N. Sutherland

Diving is one of the fastest growing sports with the New Zealand Underwater Association (PADI Franchise) now claiming that 5% of New Zealand's adult population has been either trained, or is undertaking scuba diving from training numbers and tank inspections. This figure is twice the percentage quoted from Australia of 2.5% and higher than that of other countries. In New Zealand, the numbers of both deaths and accidents requiring hyperbaric oxygen, recompression therapy, are on the increase. Last year there were 13 deaths and 35 recompression therapies (30 in Auckland) with no accurate count of the number of serious